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MicroRNA-29a Is a Friend or Foe for Cardiac Hypertrophy?

MicroRNAs (miRNAs) play an important role in the pathogenesis of structural alterations of cardiac hypertrophy (1). However, the precise mechanisms of miRNA-29a in cardiac hypertrophy are still unclear. A recent report in the *Journal* by Roncarati et al. (2) indicated that correlation with left ventricular hypertrophy parameters holds true for only miRNA-29a, which is significantly associated with both cardiac hypertrophy and fibrosis. This suggests that miRNA-29a is a potential therapeutic target for cardiac hypertrophy.

Other studies have found that down-regulation of miRNA-29a is associated with pathological cardiac hypertrophy. Abonnenc et al. (3) showed that levels of miRNA-29a were significantly reduced in a mouse model of pathological but not physiological hypertrophy (3). Furthermore, Divakaran et al. (4) indicated that Smad3 signaling plays dual roles in the heart: a beneficial role by delimiting hypertrophic growth and a deleterious role by modulating myocardial fibrosis, possibly through a pathway that entails accumulation of miRNA-29a, which decreases collagen gene expression. However, Zile et al. (5) showed that miRNA-29a levels increased 5 days after myocardial infarction and then decreased to control levels at later time points. A time-dependent change in miRNA-29a levels occurred in patients with myocardial infarction, including an early and robust increase in miRNA levels that affected myocardial fibrosis.

These findings suggest that miRNA-29a may be a good clinical biomarker for patients with cardiac hypertrophy because it seems to play a key role in the development of this condition. Additional studies with more patients and animal studies are needed to determine the precise role of miRNA-29a in cardiac hypertrophy, and therapeutic agents targeting miRNA-29a might result in innovative new therapies. All in all, we greatly enjoyed reading the article by Roncarati et al. (2) and believe that miRNA-29a may be useful for the prevention and treatment of cardiac hypertrophy.

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Pro-Inflammatory Interleukin Genotypes Potentiate Early and Advanced Atherosclerosis Differently

We read with interest the paper by Tsimikas et al. (1) in a recent issue of the *Journal*, which reported that genetic differences in the interleukin (IL)-1 gene cluster, known to be associated with inflammatory responsiveness, strongly influence the presence of